

**Cardiology**

**Neurology**

**Respiratory system**

**Abdomen**

# **Cardiology**

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### Common Cases in PACES

1. Aortic stenosis (AS)
2. Aortic Regurgitation (AR)
3. Mixed Aortic valve disease (AS and AR)
4. Mitral Regurgitation (MR)
5. Mitral Stenosis (MS)
6. Mixed Mitral valve disease
7. VSD
8. Prosthetic Valves including Metallic and Tissue Valves
9. Aortic valve replacement (AVR)
10. Mitral valve replacement (MVR)
11. Double valve replacement (DVR)
12. Mitral valve prolapse
13. Dextro-cardia
14. Heart Transplant
15. Pace Maker in situ
16. Defibrillator in situ



## 1.Aortic Stenosis

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation chart and recent ECG
- Do the Fundoscopy and Urine Analysis

I have examined this Gentleman/Lady, His/Her:

Pulse is:

- Between 70-80 / min
- Regular
- Low volume
- Slow rising

JVP is Not Raised

There is No Scar mark in the chest

Apex beat is Un-displaced and normal in Character (Pressure Loaded)

On auscultation 1<sup>st</sup> Heart sound is normal and the 2<sup>nd</sup> heart sound is soft (May be Normal)

There is a Harsh Ejection Systolic murmur heard throughout the Precordium but best audible in the aortic area that becomes Louder in expiration and radiates to the carotids.

- However there are no peripheral stigmata of infective endocarditis
- Lungs are clear to auscultation
- No peripheral edema and
- Anemia

So Based on this, my most likely diagnosis is Aortic Stenosis

**D/D:**

1. **Aortic Sclerosis** There may be an ejection systolic murmur but No Peripheral Features until quite advance then it may act like Aortic Stenosis
2. **Pulmonary Stenosis**
3. **HOCM** Murmur of HOCM increases with decreased Left ventricular filling like standing but murmur of aortic stenosis decreases on standing

**Causes (ABC-R)**

1. Aging
2. Bicuspid aortic valve
3. Congenital
4. Rheumatic fever

**Complications of Aortic Stenosis:**

1. Hemolytic anemia
2. Infective Endocarditis
3. Embolic disease from Infective endocarditis and disintegrating calcified valve
4. Heart block from calcification of AV conduction pathway
5. Heart failure
6. Sudden death

## Severity Criteria for Aortic Stenosis

### 1.Clinical

- Pulse **Low volume and Slow rising**
- **Narrow pulse pressure**
- Palpation **Heaving apex beat and Systolic thrill**
- Heart sounds **Soft or absent A2 and 4<sup>th</sup> heart sound**
- Lungs **Pulmonary HTN and Pulmonary congestion (Cardiac failure)**

### 2.Valve Area on Echocardiography

- Normal            **3-4 cm<sup>2</sup>**
- Mild AS            **> 1.5 cm<sup>2</sup>**
- Moderate AS    **1.0—1.5 cm<sup>2</sup>**
- Severe AS        **< 1.0 cm<sup>2</sup>**
- Pressure gradient **> 50 mmHg**

**Investigations:**

**1. ECG to look for:**

- Left ventricular hypertrophy (voltage criteria)
- Left ventricular strain pattern
- Left ventricular axis deviation

**2: X-ray chest to look for:**

- Calcification of the aortic valve
- Cardiomegaly
- Prominent Pulmonary vasculature due to Pulmonary HTN(If present)
- Pulmonary congestion

**3: Echocardiogram to look for:**

- Left ventricular size and function
- Aortic valve area

**4:Coronary angiography to check for:**

- Coronary artery disease and subsequent plan for CABAG

## Management of Aortic Stenosis

### 1. General measures:

- Patient's Education and explanation of the condition
- Regular follow ups to assess for progression if asymptomatic

### 2. Specific Measure:

**Medical Treatment:** Includes treatment of complications like Pulmonary Edema

**Surgical Treatment:** If Symptomatic then Valve Replacement ± CABAG

Transcatheter Aortic Valve Implantation if high risk patient (TAVI)

If Asymptomatic and following features:

- Candidate for CABAG
- Left ventricular systolic dysfunction
- Ventricular tachycardia
- Valve area < 0.6 cm<sup>2</sup>

Then **Valve replacement ± CABAG**

## 2. Aortic Regurgitation (AR)

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and Recent ECG
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is:

- Between 70-80/min
- Regular
- Good volume
- Collapsing in character

There are Prominent arterial pulsations and **JVP** is not raised

**Apex beat** is not Displaced and Thrusting in character

On auscultation **1<sup>st</sup>** Heart sound is normal and the **2<sup>nd</sup>** heart sound is soft (May be Normal)

There is an early diastolic murmur at the left sternal edge that becomes louder with the patient sitting forward and with expiration

However:

- He does not have peripheral stigmata of infective endocarditis
- Lungs are clear to auscultation
- He does not have peripheral edema and Anemia

So based on this, my most likely diagnosis is **Aortic regurgitation (AR)**

Differential Diagnosis:

1. Pulmonary regurgitation
2. Austin Flint murmur of sever aortic Regurgitation

**Causes:**

**1. Acute aortic Regurgitation:**

- Aortic dissection
- Infective endocarditis

**2. Chronic aortic Regurgitation**

- Hypertension
- Bicuspid aortic valve
- Rheumatic fever
- Aortitis e.g
  - Ankylosing spondylitis
  - Syphilis
  - Takayasu arteritis
- Connective tissue disorder (RA ,SLE etc)
- Others e.g
  - Marfan's syndrome
  - Pseudoxanthoma Elasticum
  - Ehler-Danlos syndrome

**Severity criteria for Aortic Regurgitation (Clinical)**

- Wide pulse pressure
- Third heart sound
- Increased length of diastolic murmur
- Austin flint murmur
- Pulmonary HTN
- Pulmonary congestion

## Investigations

**ECG** Usually nonspecific may show voltage criteria of left ventricular hypertrophy

**CXR** To check for:

- Cardiomegaly
- Clacification of aortic valve
- Pulmonary vasculature
- Pulmonary congestion

**Echocardiography** To look for:

- Valve morphology
- Aortic root size and dilatation
- Severity
- Left ventricular size and function

**CT/MRI** to look for:

- Aortic root and ascending aorta

**Coronary Angiography** To Look for:

- Co-existent coronary artery stenosis to make management plan better (CABAG)

**Management:**

**1. General Measures**

- Patient Education
- Regular follow ups if asymptomatic

**2. Specific measures:**

**Medical:**

- Hypertension or severe regurgitation with LV dilatation then ACE inhibitors or ARB

**Surgical**

- Valve replacement if Symptomatic and
- Pulse pressure > 100 mm Hg
- ECG Changes, LV enlargement on CXR
- EF < 50 % on Echocardiography
- Ideally replace the valve before significant left ventricular dilatation and dysfunction.

### 3.Mitral Stenosis

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is Irregular and of variable volume (Low volume if Regular)

JVP is Not raised

There are No Scar marks in the chest (There may be Left Thoracotomy scar from previous Valvuloplasty)

Apex beat is Un-displaced and Tapping in character

On auscultation 1<sup>st</sup> Heart sound is Loud and the 2<sup>nd</sup> heart sound is normal

(Pulmonary component of the 2<sup>nd</sup> heart sound may be loud)

There is a mid diastolic rumbling murmur at the apex, heard best in expiration with the patient in Left Lateral position.

However there are

- No peripheral stigmata of infective endocarditis
- Lungs are clear to auscultation
- No peripheral edema and
- Anemia

So based on this, my most likely diagnosis is **Mitral Stenosis**

D/D:

1. Austin Flint murmur of sever AR
2. Left atrial Myxoma or Thrombus

## Causes

- Rheumatic Fever
- Congenital MS
- RA
- SLE
- Carcinoid syndrome

## Investigations:

**ECG** to look for:

- Left atrial hypertrophy
- Left atrial dilatation
- AF

**CXR** to look for:

- Double right heart border
- Prominent pulmonary vasculature
- Pulmonary congestion

**TTE /TOE** to look for:

- Valve area
- Cusps mobility and calcifications
- Left atrial thrombus

**Coronary Angiography** to look for:

- Coronary artery stenosis

### Severity criteria for mitral stenosis (Clinical)

- Breathless at rest
- Low pulse pressure
- Early opening snap
- Long duration of murmur
- Signs of Pulmonary HTN
- Signs of Pulmonary congestion

### Treatment:

#### General measures

- Patient education and explanation of the condition
- Endocarditis prophylaxis
- Regular follow ups with echocardiography

#### Medical

- AF: Rate control or rhythm control strategy e.g. B-blockers, Digoxin
- Others include warfarin (According to NICE guidelines)
- and diuretics

#### Mitral Valvuloplasty if following features:

- Mobile valve
- Absent MR
- Absent thrombus

#### Surgery

- Open commissurotomy (When anatomy is not suitable for closed balloon valvuloplasty)
- Valve Replacement

#### Indications for surgery:

- Pulmonary HTN
- Pulmonary Congestion
- Hemoptysis
- Recurrent thromboembolism despite warfarin

## 4.Mitral Regurgitation (MR)

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and Recent ECG
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is Regular (or irregular) and Good volume

JVP is Not raised (May be raised)

There are No scar marks in the chest

Apex beat is Displaced (May be un-displaced) and Thrusting in character

On auscultation 1<sup>st</sup> Heart sound is Soft and 2<sup>nd</sup> Heart sound is normal

(Pulmonary component of the 2<sup>nd</sup> Heart sound may be Loud as well)

There is a pan-systolic murmur at the apex which becomes louder in expiration and radiates to the axilla

However, he does not have:

- Peripheral stigmata of infective endocarditis
- Lungs are clear to auscultation
- He does not have peripheral edema and
- Anemia

So based on this, my most likely diagnosis is Mitral regurgitation

Differential diagnosis:

- Ventricular Septal Defect (VSD)
- Tricuspid Regurgitation (TR)
- Mitral valve prolapse
- Aortic stenosis

## Causes

### 1.Acute Mitral Regurgitation:

- Infective endocarditis
- Trauma
- Rupture of chordae Tendinae

### 2.Chronic Mitral Regurgitation

- Rheumatic fever
- Mitral valve prolapse
- Infective endocarditis
- RA
- SLE
- Marfan's syndrome
- Ehler-Danlos syndrome
- Pseudoxanthoma Elasticum

### Clinical Severity Criteria:

- Soft S1
- 3<sup>rd</sup> and 4<sup>th</sup> heart sounds
- Displaced apex beat
- Pulmonary HTN
- Pulmonary congestion

### Investigation:

#### ECG to check for:

- Atrial fibrillation (AF)
- Left atrial hypertrophy
- Left atrial dilatation

#### CXR To check for

- Double right heart border
- Cardiomegaly
- Pulmonary vasculature
- Pulmonary congestion

#### Echocardiography to look for

- Anatomy of mitral valve
- Severity of MR
- Left ventricular systolic function etc

#### Coronary angiography to look for: Coronary artery disease

### Treatment:

#### General measures

- Patient education and explanation
- Antibiotics for endocarditis prophylaxis
- Annual echocardiography for assessment of progression

#### Medical treatment

- Management of AF with Rate control and anticoagulants like Warfarin
- Management of heart failure when required

#### Surgical treatment

- Valve replacement
- Patient who have symptoms despite optimum medical therapy
- Left ventricular ejection fraction < 60 %
- Left ventricular end systolic diameter > 45 mm

## 5. Ventricular Septal Defect (VSD)

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and Recent ECG
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is Regular and of Normal volume and character

JVP is Not raised

There are No Scar marks in the chest

Apex beat is Un-displaced and Normal in character

(If there is a Small defect)

- On auscultation 1<sup>st</sup> Heart sound is normal (2<sup>nd</sup> may be obscured)
- There is a Loud Pan-systolic murmur which is heard throughout the precordium but loudest at the left lower sternal edge.

However he does not have

- Peripheral stigmata of infective endocarditis
- Lungs are clear to auscultation
- He does not have edema or Anemia

So based on this, most likely diagnosis is VSD

Note:

If large defect and significant Left to Right shunting:

- Pulmonary HTN develops and it leads to a Loud P2 and the Pan-systolic murmur of VSD will become quieter.
- As pulmonary HTN increases there may be reversal of shunt from Right to Left and the development of Eisenmenger's Syndrome. In this situation, ventricular pressures will equalize and there will be a single Loud 2<sup>nd</sup> Heart sound and the VSD related murmur will disappear.

**DD:**

1. **Mitral Regurgitation**
2. **Tricuspid Regurgitaion**
3. **Aortic Stenosis**

**Investigation:**

**ECG**

**Small defects → No Changes**

**Large Defects**

- **Left ventricular hypertrophy**
- **Right ventricular hypertrophy**
- **Left atrial hypertrophy (Bifid p wave in lead II)**
- **Left atrial enlargement (Biphasic p-wave in V1)**

**CXR** In case of large defects to look for:

- **Cardiomegaly**
- **Prominence of pulmonary vasculature**
- **Pulmonary congestion**

**Echocardiography** to look for:

- **Location, size and direction of shunt**
- **Left and right ventricular size and function**

**Cardiac Catheterization** to check for:

- **Severity and reversibility of pulmonary HTN**

## Treatment

### **Small defect** with normal pulmonary artery pressure

- Reassurance
- Endocarditis prophylaxis

### **Large defect** with pulmonary HTN /Right or Left heart failure

- Endocarditis Prophylaxis
- Diuretics
- Treatment of pulmonary HTN
- VSD closure if no contraindication

## 6.Mitral Valve Replacement

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and Recent ECG
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is Normal and of Good volume (May be Irregularly irregular)

JVP is not raised

There is a midline sternotomy Scar in the chest and 2 more scars in the upper chest

Apex beat is un-displaced and Normal in character

There is a Prosthetic click that coincides with 1<sup>st</sup> Heart sound

2<sup>nd</sup> Heart sound is normal

However he (She) does not have:

- Peripheral features of infective endocarditis
- Lungs are clear to auscultation bilaterally
- He (She) does not have Anemia, Peripheral edema or Bruising

So based on this my most likely diagnosis for this gentleman (Lady) is Mitral valve replacement which is functioning very well

## 7.Aortic Valve Replacement

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is regular and of good volume

JVP is not raised

There is a mid-line sternotomy Scar in the chest and two more scars in the upper abdomen

Apex beat is un-displaced and normal in character

1<sup>st</sup> Heart sound is normal but there is a prosthetic click that coincides with the 2<sup>nd</sup> heart sound

However he (She) does not have

- Peripheral features of infective endocarditis
- Lungs are clear to auscultation bilaterally
- He (She) does not have anemia, peripheral edema or any Bruising

So based on this, my most likely diagnosis for this gentleman (Lady) is Aortic valve replacement which is functioning very well

## 8. Dual Valves Replacement

I would like to complete my examination by:

- Taking Detailed History
- Check the Observation Chart and Recent ECG
- Do the Fundoscopy and Urine Analysis

I have Examined this Gentleman/Lady, His/Her:

Pulse is regular and of good volume

JVP is not raised

There is a midline sternotomy Scar in the chest and two more scars in the upper abdomen

Apex beat is un-displaced and normal in character

There are prosthetic clicks which coincide with 1<sup>st</sup> and 2<sup>nd</sup> Heart sounds

However he (She) does not have:

- Peripheral features of Infective endocarditis
- Lungs are clear to auscultation bilaterally
- He (She) does not have anemia, peripheral edema or any Bruising

So based on this my most likely diagnosis for this gentleman (Lady) is Dual Valves Replacement which are functioning very well.









# Neurology

## 1.Idiopathic Parkinson's Disease

**Parkinsonism** is a general term for Movement disorder which means any combination of  
(**BRITish Gentleman**)

- **Brady-kinesia**
- **Rigidity**
- **Postural Instability** and
- **Resting Tremors**

Parkinsonism has many causes but idiopathic Parkinson's disease is the most common

**Other causes** of Parkinsonism include:

- Drugs e.g Neuroleptic --may be reversible
- Parkinson's Plus Syndromes
- Wilson's disease
- Post Encephalitic
- Brain anoxia
- Lewy body dementia
- Toxins induced

### **Idiopathic Parkinson's Disease:**

- A neuro-degenerative disorder that begins between 45 to 65 years of age
- Characterized by an imbalance between the neurotransmitters Dopamine and Acetylcholine due to dopamine depletion
- So treatment of motor disturbance is focused at readdressing this disorder by giving anti-cholinergics or dopamine agonists
- Slowly progressive disorder
- Asymmetrical involvement is the hall mark though in advanced cases all the limbs may be affected
- Dementia is usually the late feature of Parkinson's disease
- Seborrhea of scalp and face may be noted as well
- Head titubations are not present (May be present in Benign essential tremors)
- Tremors may be absent in 20 % of cases
- Asymmetrical foot-tapping tremors may also be present
- Tremor may be present about the mouth and lip
- If gait is wide based think of cerebellar involvement as well ---Parkinson's plus syndrome

### **Parkinson's Plus syndrome:**

**Neuro-degenerative disorders with features of Parkinsonism and other neurological involvements**

#### **May have:**

- Symmetrical involvement
- Early onset dementia
- Early falls
- Postural hypotension
- Pyramidal signs (not explained by stroke)
- Cerebellar signs
- Poor response to Levodopa

**Parkinson's Plus syndrome has 3 components:**

**1. Progressive Supra-nuclear Palsy**

- Bradykinesia
- Rigidity
- Supra-nuclear disorder of eye movements and Pseudo-bulbar palsy

**2. Multiple system atrophy**

Autonomic insufficiency may be accompanied by:

- Parkinsonism
- Pyramidal deficits
- Lower motor neuron signs and cerebellar dysfunction

**3. Cortico-basal degeneration**

- Asymmetrical parkinsonism
- Signs of Cortical dysfunction like Apraxia, Sensory inattention, Dementia and Aphasia

**Commands:**

- Look at the patient and proceed
- Look at the face and proceed
- This gentleman has presented with tremors in the Hands, Do the neurological examination as appropriate
- This gentleman has difficulty in walking, Do the neurological examination as appropriate

### Examination scheme:

#### Introduction

- Assess speech---Low volume, monotonous and slurred

#### General inspection

Look at the **Face** carefully

- Expressionless
- Mask like
- Drooling saliva
- Infrequent blinking etc

Ask the patient to keep the **hands in semi-prone position** --Resting tremors

#### Hands

- Tremors
- Brady-kinesia
- Finger Nose testing
- Rigidity--cog-wheel

#### Elbows

- Rigidity---lead pipe (make use of Synkinesis)

#### Face and Eyes

- Can you Plz close your eyes gently—Blepharclonus
- Eye movements—Nystagmus and Upward gaze palsy

**Gait:** can you Plz stand up and walk few steps

- Freezing
- Posture
- Arm swinging
- Turning back

**Hand writing**

- **Micrographia**

**Functional capacity**

- **Button and unbutton**

**I would like to complete my examination by taking**

- **Detailed history including history of medicines**
- **Complete neurological examination**
- **MMSE**
- **Check the postural drop**

**Recording:**

Well,

I have examined this gentleman

He has expressionless face with low volume, monotonous speech and infrequent blinking

**In the right upper limb, he has evidence of: (If Right upper Limb is involved)**

- Pill rolling resting tremors
- Bradykinesia
- Rigidity being cogwheel at the wrist and lead pipe at the elbow which increases with synkinesis

**When asked to walk** He has difficulty in starting his walk

**Once he started his walk**

- He adopts a stooped posture and walks with narrow based shuffling gait and has reduced arm swinging on the right side
- He also has difficulty in turning back
- He also has evidence of Micrographia and problem with functional capacity in the form of buttoning and unbuttoning

However he does not have evidence of upward gaze palsy or cerebellar involvement

So based on this and because of asymmetrical signs

My most likely diagnosis for this gentleman is **Idiopathic Parkinson's disease**

### Differential Diagnosis:

1. **Benign essential tremors** if old age and presented with tremors
2. **Other movement disorder like Huntington Disease**---may have family history and dementia
3. **Wilson's disease**---may have other features like early onset, chronic hepatitis with abnormal movements etc
4. **Depression**--- with expressionless face may be associated with disease itself

### Investigations:

Diagnosis is clinical.however:

- **Brain imaging** like CT and MRI done to rule out structural pathology
- **DaTSCAN using SPECT** to differentiate Parkinson's disease related tremors from Benign essential tremors
- **Psychometric testing** if symptoms of cognitive impairment
- **Serum Urate level** as a Prognostic indicator—as the level rises the rate of progression declines

**Treatment:** Treatment is generally symptomatic

**A. General measures:**

1. Patient's education and explanation of the condition
2. Education of the family and care-givers
3. MDT package with aim for physical and speech therapy
4. Simple measures to improve daily living with the help of occupational health physician like:
  - Placement of rails
  - Special bath seats
  - Cutlery with long handles
  - Non-slip rubber table mats
  - Shirt without buttons
  - Shoes without laces
  - Fixing the rugs to avoid falls
  - Pendant alarm system to call for help if required
  - Devices to amplify the voice etc

**B. Medical Treatment**

Generally not required early in the disease course

1. Amantadine

2. Dopaminergic drugs

a. Levodopa with Carbidopa

b. Dopamine agonists like Pramipexole and Ropinirole

Others include: Bromocriptine, Pergolide and Cabergoline etc and

Novel delivery system in the form of Transdermal patch have also been developed like Rotigotine

c. Selective Monoamine Oxidase B inhibitors (MAO-B inhibitors) like Selegiline and Rasagiline

3. COMT Inhibitors like Entacapone and Tolcapone

**4.Anticholinergic Drugs** like Benzatropine,Benzhexol,Procyclidine etc

**5.Apomorphine** Given subcutaneously if patient can't tolerate oral medicines

**6.Antipsychotics:** Clozapine,Olanzapine,Quetiapine and Risperidone etc for side effects of therapy

### C. Brain Stimulation

### D. Other Surgical approaches

- Thalamotomy
- Pallidotony
- Sub-thalamotomy

### E. Gene Therapy

## B. Medical Treatment detail: (Optional)

Generally not required early in the disease course

### 1.Amantadine

- Mode of action is unclear
- May be given for mild symptoms without disability
- It also improves Dyskinesias resulting from chronic Levodopa therapy
- **Dose:** 100 mg BD

### 2.Dopaminergic drugs

#### a. Levodopa with Carbidopa

- Levodopa is converted to dopamine
- Much effective for Motor symptoms
- Response fluctuations may occur like On--Off phenomenon
- Nausea,vomiting,hypotension may occur initially
- Restlessness,confusion,dyskinesias may occur later

**Dose:**

- Started with small dose
- Brand name available is **Sinemet**—Carbidopa:Levodopa
- 2 strengths are available
- Tablet Sinemet 25/100 (25mg Carbidopa/100mg Levodopa) may take 3 times daily initially and may gradually increase depending upon the response
- Tablet sinemet 25/250 (25mg Carbidopa/250 mg Levodopa)

### To reduce fluctuations

1. Controlled release preparations may be used
2. Carbidopa/Levodopa/Entacapne combination (**Stalevo**) can be given
3. Protein should be taken at minimum recommended levels and last meal could be taken at night as the last meal

### b.Dopamine agonists:

**Pramipexole and Ropinirole**---act on dopamine receptors

- Effective both in early and late disease
- Lower incidence of Response fluctuations and dyskinesias
- Can be given before start of Levodopa
- Can be given with low dose of Sinemet 25/100 TDS when treatment is started initially
- In above case the dose of Sinemet can be kept constant but agonist dose can be increased gradually

Dose:

**Pramipexole :**

- Starting 0.125 mg TDS can be doubled every week until between 0.5 to 1.5 mg TDS and response is gained

**Ropinirole:**

- Starting dose is 0.25 mg TDS
- Maximum between 2 and 8 mg TDS daily

Others include:

**Bromocriptine, Pergolide and Cabergoline etc**

Novel delivery system in the form of Transdermal patch have also been developed like **Rotigotine**

### c.Selective Monoamine Oxidase B inhibitors (MAO-B inhibitors)

- They inhibit metabolic breakdown of Dopamine
- Can be used as adjunctive therapy in patients with response fluctuations with Levodopa
- **Selegiline** —5 mg orally with breakfast and lunch
- **Rasagiline**—1 mg daily in the morning

### 3.COMT Inhibitors:

- Reduce the metabolism of Levodopa
- Can be used as an adjunct to Levodopa/Carbidopa in patients with response fluctuations or inadequate response
- Entacapone 200 mg with each dose of Sinemet
- Tolcapone 100 mg or 200 mg TDS daily with Sinemet
- With above medicines the dose of Sinemet can be reduced by upto one-third concurrently with every dose
- With Tolcapone LFTs are performed 2 weekly for first year
- Stalevo combination of Levodopa/Carbidopa/entacapone

### 4.Anticholinergic Drugs

- Helpful for Tremors and rigidity but less so for bradykinesia
- Side effects limit routine use
- Avoided if cognitive impairment
- Elderly have poor tolerance

Examples:

Benztropine, Benhexol, Procyclidine etc

### 5.Apomorphine given subcutaneously

### 6.Antipsychotics:

- Confusion and psychotic symptoms because of side effect of therapy can be managed with atypical antipsychotics like Clozapine,Olanzapine,Quetiapine and Risperidone etc

### C. Brain Stimulation:

High frequency stimulation of the sub-thalamic nuclei or golbus pallidus internus

Deep brain stimulation

**D. Other Surgical approaches**

- **Thalamotomy**
- **Pallidotomy**
- **Sub-thalamotomy**

**E. Gene Therapy**

## 2. Myotonia Dystrophica

A Multi-system disorder which may cause:

- Slowly progressive muscle wasting
- Myotonia
- Cataracts
- Cardiac conduction defects
- Endocrine deficiencies
- Respiratory problems

Autosomal Dominant disorder caused by Trinucleotide Repeat Expansion (CTG)

So there is earlier and severe presentations in the successive generations

It has 2 types

Type 1 is classical form

Type 2 has similar features to type 1 except there is proximal weakness and wasting

Commands:

- Look at the face and examine the hands
- Look at the patients and examine the appropriate neurological system
- This patient has family history of complication from anaesthesia, do the appropriate neurological examination
- This patient has presented with weakness in the hands, do the appropriate neurological examination
- This patient has presented with collapse, do the appropriate neurological examination (Heart block)
- This gentleman/Lady has presented with dysphagia, do the relevant neurological examination

**Examination scheme:**

**General examination:**

Look for clues like:

- Middle aged male or female with thin face
- Sleepy look
- Pace maker
- Foot Orthoses nearby

**Face (On inspection)**

Frontal baldness (if male and may be wearing a wig)

Wasting of facial muscles (Thinning of face sometimes not much marked)

Bilateral ptosis (Less often may be unilateral)

Mouth hanging open (Sleepy appearance)

Can you Plz close your eyes tightly? Now plz open your eyes. (Myotonia)

Can you Plz whistle? (Perioral weakness)

**Face, Eys, Neck (Palpation)**

I am going to check power of your eyes and face muscles, is that ok with you?

Ok ,Plz close your eyes and don't let me open them?

Thank you!

Now Plz clench your teeth?

Ok, Now I am going to check power of your neck muscles.

Plz turn your face towards opposite side and push against my hand.

(Feel for sternocleidomastoid muscle)

Thank you!

Now Plz push your face to other side against my hand.

Thank you!

Now I am going to throw light into your eyes. Is that ok with your? (**Cataracts**)

Ok thank you!

Now I am going to examine your hands and arms. Is that Ok with you?

**Hands (Inspection and palpation)**

- Wasting and pin prick marks
- Delayed grip (**Usually checked on hand shaking during introduction**)
- Percussion Myotonia
- Power of hand muscles
- Brachioradialis reflexes

**Inspection of Feet for wasting and Gait for high steppage**

I would like to complete my examination:

- By taking detailed history
- Do the Fundoscopy (Diabetic Retinopathy)
- Thyroid examination (Goiter or May be scar)
- Heart and chest examination (Atrial Fibrillation, Pace maker, CCF etc)
- Dip the urine for glycosuria
- And check for Testicular atrophy

**Recording for Myotonia Dystrophica:**

Well,

I have examined this Gentleman/Lady

He/She has:

Thin, elongated, expressionless face with frontal baldness (**If male**)

With bilateral ptosis and difficulty in opening the eyes after firm closure along with Loss of red reflex in the eyes (**Because of Cataracts**)

There is wasting and weakness of facial muscles

There is also wasting and weakness of neck muscles as well

**On examination of the Hands:**

There is wasting and weakness of hand muscles with Pin prick marks of the fingers

There is also evidence of delayed grip and Percussion Myotonia

The deep tendon Reflexes are depressed

He/She also has high steppage gait

So based on this, My most likely diagnosis is **Myotonia Dystrophica**

### Differential Diagnosis:

#### DD of Myotonia:

- Myotonia Congenita
- Mild Tetanus

#### DD of Distal weakness:

- Peripheral Neuropathy
- HMSN
- Distal Spinal Muscular Atrophy
- Inclusion body Myositis

### Investigations:

Diagnosis is clinical one but some tests can be done as supportive measures and to look for underlying associated complications

#### Blood tests:

- Creatine Kinase (CK)
- FSH(May be raised)
- Teststerone (May be Low/Normal)
- Fasting Glucose and Hb A<sub>1c</sub>

#### Other tests include following:

- ECG and Echocardiography
- EMG
- Genetic Testing and Counselling

**Treatment:**

- There is no cure
- Treatment is symptomatic

**General Measures:**

- Patient's education and explanation of the condition
- Education of the family as well
- Avoidance of certain factors like Opiates, Statins, and care about Anaesthetic agents

**MDT care package like:**

- Physiotherapist for muscle weakness
- Occupational health physician for wrist splints, foot orthoses and other necessary measures and aids at home if required
- Help with Speech and language therapy
- Eye specialist if cataracts or retinopathy
- Cardiologist if heart involvement

Patient may be guided about Myotonia Dystrophy Support Group

**Medical treatment:**

**Myotonia--Phenytoin or Procainamide**

**GI symptoms:**

**Delayed gastric emptying--Metoclopramide**

**Small intestinal bacteria overgrowth—Antibiotics**

### 3. Myasthenia Gravis

**Myasthenia Gravis is an autoimmune disorder characterized by autoantibodies binding to acetylcholine receptors resulting in variable degree of block of neuromuscular transmission.**

**It may occur at all ages and may be associated with other autoimmune conditions.**

**It is most common in young women (Likely Young lady in PACES)**

**It may be unmasked by recent infection**

**It may also be exacerbated before menstruation.**

**It may present with fluctuating weakness of commonly used voluntary muscles.**

**Patient may present with:**

- Diplopia
- Problem with chewing or swallowing
- Respiratory problems or weakness of limbs
- Weakness may be localized in the form of ocular Myasthenia Gravis or Generalized
- Most commonly Extra-ocular and other cranial muscles may be affected
- Most important being the diurnal variations

**On clinical examination there may be weakness and Fatigueability**

- Ptosis (commonly asymmetric) with normal pupillary reflexes
- Sensations are normal
- Reflexes are not affected

**Don't forget Myasthenic Crisis with Respiratory problems which may require admission in ICU and ventilatory support**

**Commands:**

- This Lady has presented with recurrent falls, do the appropriate neurological examination
- This Lady has presented with double vision, examine her.
- This Lady has presented with fatigue , do her neurological examination
- This Lady has presented with Dysphagia , do the appropriate neurological examination

**Examination Scheme:**

**General inspection:**

**Young lady (Most likely)**

**Thymectomy scar or Mid-line Sternotomy scar (May be present which is a big clue)**

**There may be evidence of other autoimmune disorders as a clue like RA,Thyroid problem,Vitiligo etc**

**Jaw supporting sign**

**Cushingoid appearance (May be present)**

**Immunosuppression stigmata (May be present)**

**Face and Eyes (Ptosis—may be unilateral or asymmetric bilateral with furrowing of forehead and normal pupils)**

**Fatigueability in Eyes**

**Brief Rest of Eyes (Improvement)**

**Eye Movements (Diplopia)**

**Power of Eye muscles**

**Now Plz clench your teeth (Temporalis and masseters)**

**Power of jaw muscles (Especially jaw closing)**

**Plz puff out your cheeks**

**Smile (Myasthenic Snarl)**

**Now can you Plz open your mouth and Say ahh! (Movement of uvula)**

**Ok thanks**

**I would like to complete my examination by taking detailed history including history of medicines**

**Look for thymectomy scar (If not exposed during examination)**

**Will look for other autoimmune associations**

**Recording:**

Well, I have examined this Lady:

She has evidence of :

**Thymectomy Scar (Midline sternotomy scar)**

**Vitiligo etc**

**There is evidence of:**

- Variable Ptosis accentuated by sustained upward gaze and it Improves with brief eye closure
- Weakness of eye muscles with evidence of diplopia in multiple directions
- Bilateral facial muscle weakness with lack of facial expression and horizontal smile  
**(Myasthenic Snarel)**
- jaw weakness
- Voice has nasal twang with decreased movement of Uvula

**Weakness of neck Muscle**

**On examination of the upper Limbs, there is evidence of**

- Proximal weakness and Fatigueability
- Deep Tendon Reflexes and sensations are normal

**Gait**

**Differential Diagnosis:**

- 1.Lambert Eaton Myasthenic Syndrome
- 2.Primary myopathies like inflammatory and mitochondrial myopathies
- 3.Miller-Fisher syndrome
- 4.Snake bites
- 5.Botulism
- 6.Drug induced Myasthenia like syndrome e.g D-Penicillamine

**Investigations:**

- 1.Ice pack test
- 2.Tensilon Test
- 3.Serology like anti-AChR antibodies and anti-MuSK antibodies
- 4.Electrophysiology studies to see Repetitive Nerve stimulation (RNS) and Single fiber electromyography (SF-EMG)
- 5.CT of the Chest

**Treatment:**

**General treatment:**

- Patient's education and explanation of the condition
- Education of the family
- MDT care package if required
- Vaccinations including Pneumococcal and H influenza
- MidcAlert bracelets
- Help with Support Groups
- Check the Acetylcholine receptors antibodies annually

**Medical Treatment:**

**1.Cholinesterase Inhibitors: e.g Pyridostigmine**

**2.Immunosuppressive therapy: Corticosteroids and Others include Azathioprine,Mycophenolate Mofetil,Ciclosporin or Tacrolimus**

**Treatment of complications:**

**1.Myasthenic Crisis:** severe weakness affecting Respiratory Function and require intubation and ventilatory support can be triggered by infection,weaning of immunosuppression,surgery or pregnancy etc

**Treatment:**

- Admission to ICU
- Cholinesterase inhibitors are usually stopped to prevent excessive secretions in airways
- IVIG or Plasmapheresis
- Steroids
- Antibiotics

**2.Cholinergic Crisis:**

- Because of excess treatment
- Can cause weakness
- Treated by reducing the dose

**3.Pneumonia especially aspiration pneumonia** and is Treated accordingly

**Surgical Treatment:**

- **Thymectomy**
- **Absolute indication is presence of Thymoma**
- **All Myasthenia Gravis patients with age less than 55 years are treated with Thymectomy**
- **Benefits of surgery may be apparent after many years**

## 4.Cerebellar Syndrome

### Commands:

- This Gentleman/Lady has presented with sudden balance problem, do the appropriate neurological examination
- This Gentleman/Lady has presented with walking difficulty, do the appropriate neurological examination

### Examination scheme

#### General Inspection

#### Hands & Arms

- Intention Tremor
- Dysdiadochokinesia
- Finger nose testing
- Tone

#### Eye movements (INO if cause is MS and Nystagmus)

#### Speech

#### Gait (May be checked at the start)

#### Balance with open eyes and close eyes (To differentiate from sensory ataxia as well)

#### Walking if feasible

Further examination scheme for cerebellar Lesion includes the following:

1. **Examination of 5<sup>th</sup>, 6<sup>th</sup>, 7<sup>th</sup> and 8<sup>th</sup> Cranial Nerves (CP-angle Syndrome)**
2. **Evidence of Stroke**
3. **Eyes and Tongue (Jaundice and Glossitis for the underlying B-12 deficiency)**
4. **Lymph Nodes (Paraneoplastic)**
5. **Thyroid (Hypothyroidism may cause cerebellar involvement)**
6. **Other features of Malabsorption (Vit. B 12 and Vit. E deficiency)**
7. **Autoimmune features like Vitiligo (Autoimmune Gastritis leading to B 12 deficiency)**
8. **Fundoscopy (Papilledema for posterior cranial fossa tumor involving cerebellum)**
9. **Rest of the causes on History e.g Anti-epiletics, Alcohol, familial (Ataxia telangiectasia)**

**Recording for Imaginary cerebellar Lesion on the Right side:**

Well, I have examined this Gentleman/Lady:

**He is ataxic on the right**

**In the right upper Limb there is evidence of:**

- Intention tremor
- Dysdiadochokinesia
- Impaired finger nose testing with evidence of dysmetria
- Hypotonia

**There is Nystagmus in the eyes with fast component towards the right side**

**The speech is slurred and explosive**

**In the right lower limb, there is impaired heel shin test**

**He is ataxic on the right side, with broad based gait**

### Causes:

Common causes include **Alcohol, MS ,Stroke and Drugs** but following is the list

- Demyelination (**MS**)
- Friedrich's ataxia
- Parkinson's plus syndrome (**Multiple system atrophy**)
- PICA or Brain stem Stroke (**Contralateral weakness**)
- Posterior crania fossa tumor
- Paraneoplastic (**Bilateral ---anti – Yo and anti-Hu antibodies may be present**)
- Hypothyroidism
- Vitamin B 12 deficiency
- Vitamin E deficiency and Abetalipoproteinemia leading to vitamin E deficiency
- Drugs like Phenytoin, Carbamazepine etc
- Familial like Ataxia telangiectasia
- Alcoholic cerebellar degeneration
- Wilson's disease

### Differential diagnosis:

- Sensory ataxia
- Vestibular Lesions

**Investigations:**

Can be done according to underlying possible cause on clinical basis e.g

- LFTs
- Thyroid function tests
- Parathyroid hormone
- Vitamine E and Vitamin B 12 level
- Serum ceruloplasmin
- Autoantibody screening especially for paraneoplastic

**Brain imaging for conditions like**

- MS
- SOL
- Stroke

**Treatment:** Depends on the underlying cause

## 5. Recordings of Peripheral Neuropathy

### A. Peripheral Sensorimotor Neuropathy

I have examined this gentleman (**Lady**)

In the Lower parts of the Legs, he (**She**) has evidence of:

- Dry and shiny skin with loss of hair upto the mid-calves
- Ulcers and callosities on the sole of the feet
- Ankle joints are Swollen with reduced range of movements (Charcot joints if present)

There is also wasting and Lower Motor Neuron type weakness in this part as well as evidenced by:

- Hypotonia
- Hyporeflexia and
- Downgoing Plantars

There is also bilateral symmetrical sensory loss in a stocking distribution as evidenced by:

- Impaired pin prick sensation
- Impaired joint position sensation

He (**She**) has wide based and high steppage gait with ataxia with positive Romberg Test

So based on this my most likely diagnosis is Peripheral Sensorimotor Neuropathy

## B. Peripheral Motor Neuropathy

I have examined the Lower Limbs of this gentleman (**Lady**)

He (**She**) has Lower Motor Neuron type weakness in the distal parts of Lower Limbs as evidenced by:

- **Wasting**
- **Hypotonia**
- **Hyporeflexia and**
- **Downgoing reflexes**
- **High steppage gate**

However the sensations are normal

So based on this my most likely diagnosis is Peripheral Motor Neuropathy

### C. Peripheral Sensory Neuropathy

I have examined this gentleman (Lady), He (She) has evidence of:

- Shiny and dry skin with Loss of hair upto the mid-calves
- Ulcers and callositie on the soles of feet
- The ankle joints are swollen with abnormal range of movements(Charcot joints)
- Bilateral symmetrical sensory loss in a stocking distribution with impaired pin prick and joint position sensations

Gait is wide based and there is ataxia with positive Romberg test

However, there is no wasting and weakness and reflexes are not depressed

So based on this, my most likely diagnosis is Peripheral Sensory Neuropathy

**Causes of Sensorimotor neuropathy:**

- Alcohol
- Diabetes
- Hypothyroidism
- Uremia
- Vasculitis
- HMSN

**Causes of Sensory Neuropathy:**

- Alcohol
- Diabetes
- Hypothyroidism
- Uremia
- Vasculitis
- Vitamin B 12 Deficiency
- Drugs
- Causes of Motor Neuropathy:
  - Lead
  - Diphtheria
  - Porphyria
  - Drugs

**Investigations:**

**General investigations:** Simple blood tests to Look for possible cause like:

- CBC,ESR,CRP
- LFTs
- RFTs
- Serum Electrolytes
- Fasting Blood Sugar

**Other specific blood Tests like:**

- Thyroid Function Tests
- Serum B 12 and Folate level
- Serum protein electrophoresis
- Autoimmune profile
- Infectious screening: Like HIV,Syphilis and Lyme's Disease
- 24 Hour Urine analysis: For Porphyrias and Bence Jones Proteins
- CSF analysis:For GBS and CIDP

**Nerve conduction studies and EMG sometimes**

**Genetic Testing for HMSN**

**Nerve biopsy**

**Treatment:**

**General Measures:**

- Patient's education and explanation of the condition
- MDT care package
- Education about feet care to prevent complications like Charcot's Joint, ulcers etc
- Prevention of risk factors like alcohol

**Medical treatment:**

Depends upon underlying possible cause e.g IVIG for GBS

Diabetic Neuropathy with Tight glycemic control with Diet, Exercise, Drugs and insulin etc

**Medicines:**

**Painful Sensory neuropathy:**

- Pregabalin
- Gabapentin
- Duloxetine
- TCA
- SSRI

**Treatment of Autonomic Neuropathy:** This is symptomatic

**Orthostatic Hypotension:**

- Simple measures like avoiding sudden changes in posture
- Avoidance of Medicines that aggravates hypotension
- Raising the head end of the bed by 45 Degrees
- Fludrocortisone

**Gastroparesis:**

- Frequent small meals
- Metoclopramide
- Erythromycine

**Bladder Dysfunction:**

- Bethanechol
- Bladder training or catheterization if sever problem

**Erectile dysfunction:**

- PDE-5 inhibitors e.g Sildenafil

## 6. Spastic Paraparesis

- Pyramidal pattern of weakness in the Lower Limbs
- It can be acute (Sub-acute) and chronic and
- It can be compressive and non-compressive
- In compressive type spastic Paraparesis, there will be no signs above the Lesion
- Urinary catheter may be a clue that Spinal cord is involved

### Commands:

- Examine the gait and then proceed as appropriate
- Examine the Legs

### A.Spastic Paraparesis with Normal Sensations

1. **MND** (Fasciculations in the lower limbs may be a clue)
2. **HSP** (Upper Limb examination may be normal)
3. **Para Sagittal Tumor** (May have Cortical sensory loss e.g Loss of both Stereognosis and Graphasthezia)

Examination:

- **Motor System**
- **Coordination**
- **Sensory System**
- **Gait**

Then following

- **Eye movements and Tongue (Fasciculations etc)**
- **Fundoscopy (Papilledema)**

## **B.Spastic Paraparesis + Sensory Level I.e Absence of signs above the level**

**Compressive Lesion due to**

1. Tumor
2. Trauma
3. TB
4. Abscess
5. Hematoma
6. Disc Herniation

**Examination:**

- **Motor System**
- **Coordination**
- **Sensory System**

**Then following**

- **Back of spine (Scar,Gibbus,Deformity,Swelling etc)**

### C.Spastic Paraparesis + Loss of Pain Sensations

(Spinothalamic tracts in upper Limbs are affected but Dorsal Column is Normal i.e Dissociated Anaesthesia)

**1.Syringomyelia** (Wasting of the hands may be a clue with impaired pain sensations in the upper limbs as the Syrinx usually involves the cervical cord and it may extend into the brain stem as well)

**2.Anterior spinal artery stenosis**

#### Examination:

- **Motor System**
- **Coordination**
- **Sensory System**

Then following examination

- **Hands (Wasting, Burn marks etc)**
- **Pulse (Irregular)**

D.Spastic Paraparesis + Impaired joint position sensation (Dorsal column involvement)

**C- MAST**

1. **Cervical myelopathy** (Cervical collar may be a clue)
2. **Multiple sclerosis** (Dysarthria,Nystagmus and cerebellar signs in upper limb may be a clue)
3. **FA** (Dysarthria, Nystagmus and Cerebellar signs in upper limb and bilateral Pes Cavus may be clues)
4. **SACD**
5. **Taboparesis**

**Examination:**

- **Motor System**
- **Coordination**
- **Sensory system**

Then examination of eyes for the following features:

- **Jaundice** (Others tongue for Glossitis and splenomegaly for Vit. B-12 related features)
- **Nystagmus** (Cerebellum in MS)
- **Argyll Robertson pupils** (Syphilis)

## E.Spastic Paraparesis + Depressed Ankles

### P-MAST

1. Peripheral Neuropathy + **Cervical myelopathy or Bilateral stroke**
2. MND
3. FA
4. SACD
5. Taboparesis

### Examination:

- **Motor System**
- **Coordination**
- **Sensory system**

Then following scheme

- **Jaundice** (Others tongue for Glossitis and Splenomegaly for Vit. B-12 related features)
- **Argyll Robertson pupils** (Syphilis)
- **Eye movements and tongue** for fsciculations(MND)

## 6.Others

- **Tropical spastic Paraparesis (HTLV-1 associated Myelopathy)**
- **Transverse Myelitis (causes may be Viral,Bacterial,Demyelination,Radiation,Vascuilitis etc)**  
  
Inflammation of the cord which may be diffuse at one or more levels and may affect all the spinal cord function which may result in bilateral Motor,Sensory and Sphincter deficit below the level of the lesion.

**Investigations:**

- With acute or sub-acute onset following investigations are recommended
- FBC,ESR,CRP
- Chest X Ray
- CT Myelogram or MRI of the spine

**With Non-acute presentation following investigations should be carried out**

**Blood tests e.g**

- CBC,ESR,CRP
- Autoantibody screening
- Vitamin B 12 and Folate level
- Infectious screening with serological tests for HIV,Syphilis ,HTLV-1
- Blood cultures,sputum cultures and early morning urine

**Lumbar puncture with CSF analysis**

**MRI of the brain**

**Biopsy of the mass if required**

**EMG and NCS as appropriate**

**Management:**

**Depends upon underlying cause, Like for compressive Lesions we can proceed as below**

- MDT care package including Physiotherapist, Occupational health physician, Neurophysician and Neurosurgeon etc
- If acute or subacute onset, urgent neurosurgical opinion
- Acute onset but no sphincter involvement yet, Urgent surgical decompression
- If malignant compression, IV dexamethasone and Spinal radiotherapy may be required
- Other medical measures include Muscle relaxants like Baclofen, Tizanidine and Dantrolene etc
- Neuropathic pain can be treated with TCA, Pregabalin and gabapentin etc

## Recording for Upper Motor Neuron Lesions

I have examined this gentleman (Lady).

- **He (She) has Upper Motor Neuron weakness in Lower Limbs as evidenced by:**
- **Hyper-tonia**
- **Hyper-reflexia**
- **Up-going plantars**

Other possible features depending upon the cause like:

- **He has impaired Heel shin test in the Lower Limbs**
- **There is impaired Pin Prick sensation in the Lower Limbs with sensory Level upto T-10**
- **There is also evidence of impaired joint position sensation in the Lower Limbs**
- **He (She) has wide based (Dorsal column or Cerebellum) and high steppage gait (If Foot drop)**
- **The Rombergs test is also positive (Gait is ataxic if cerebellum is involved)**

## Recording For Lower Motor Neuron Lesions

I have examined this gentleman (Lady)

He (She) has Lower Motor Neuron weakness in the Lower Limbs as evidenced by:

- Wasting and fasciculations
- Hypotonia
- Hyporeflexia and
- Downgoing plantars

Other features may also be present depending upon the cause e.g if MND then may be following:

- Dysarthria with Nasal quality speech
- Wasting and fasciculations in the tongue
- Palatal paralysis

## Recording for Combined Upper and Lower Motor Neuron weakness

(e.g Motor Neuron Disease)

I have examined this gentleman (Lady)

He (she) has both Upper and Lower Motor Neuron features in the Lower Limbs as evidenced by:

- Wasting and fasciculations (May be the only features of LMN Lesion in the Lower Limbs)
- Hyper-tonia
- Positive ankle clonus
- Hyper-reflexia
- Upgoing plantars

But with

- Normal sensations

And other features may be present like:

- Dysarthria with Nasal quality speech
- Wasting of tongue with fasciculations
- Palatal weakness

Other relevant examination may include:

- Eye Movements
- Examination of Tongue for wasting and fasciculations (It may also be spastic if Pseudo-bulbar)

Examination of Upper Limbs may show wasting and fasciculations

## 7.Charcot Marie Tooth Disease (**CMDT**) or HMSN

**Most common inherited neurological disorder**

**Patient may be young**

**Foot orthoses ankle supports etc (Foot drop due to distal wasting)**

**Bilateral Pes Cavus and Hammer toes (in long standing cases)**

**Tendon release scars** may be present

**Distal wasting and fasciculations**

**Hereditary Peripheral Neuropathy with predominant Motor involvement**

**Dorsal column may also be affected (Impaired joint position)**

**Some times there may also be impaired Pin-prick sensation in the stocking distribution**

**Nerves may be palpable**

## Recording for Charcot-Marie-Tooth Disease

I have examined the Lower Limbs of this gentleman (**Lady**)

He (She) has evidence of Bilateral Pes-cavus (**Scar Marks etc**)

There is evidence of distal wasting (**Giving inverted champagne bottle appearance**)

In distal parts of Lower Limbs, there is Lower Motor Neuron weakness as evidenced by:

- Wasting and Fasciculations
- Hypotonia (May be normal)
- Depressed ankles
- Downgoing plantars
- **Palpable Nerves** (May be--Particularly at the medial malleolus)
  
- There is impaired Pin-prick sensation in the stocking distribution (May be normal)
- Impaired joint position sensation and vibration (May be present)
  
- He (She) has wide based and high steppage gait and ataxic with positive Romberg test

## Recording of CMTD (**HMSN**) in Upper Limbs

There is evidence of Pseudo-athetosis (**When asked to close the eyes by stretching the arms**)

Lower motor Neuron type weakness in the distal parts of Upper Limbs as evidenced by:

- Wasting of hands and evidence of Fasciculations
- Hypotonia (May be normal)
- Hyporeflexia
- **Palpable nerves** (Especially Ulnar nerve at the elbow)
  
- Impaired pin prick sensation in the glove distribution (May be normal)
- Impaired joint position and vibration sensation
  
- Gait is wide based, High steppage and ataxic with positive Romberg's test

### DD:

- Peripheral Neuropathy due to other causes
- Mononeuropathy
- L4-5 Nerve root Lesions

### Investigaions:

- NCS
- Genetic studies

**Treatment:**

**General Measures:**

- Patient's education and explanation of the condition
- MDT care package involving Physiotherapist, Neurophysician, Orthopedic surgeon and Occupational health physician
- Avoidance of triggers for neuropathy
- Support groups
- Genetic counselling

**Medical treatment:**

No cure

Treatment is symptomatic

## 8.Friedreich's Ataxia

- Visual Aids
- Hearing Aids
- Pace maker
- Foot Orthoses

**Bilateral Pes Cavus and cerebellar Signs are highly suggestive of Friedreich's ataxia**

### Others

- Kyphoscoliosis
- High arched palate

### Features in the Lower Limbs:

**Corticospinal tracts** Pyramidal weakness,extensor plantar responses

**Peripheral neuropathy** Long standing leading to Bilaterla Pes cavus,hammer toes,Distal wasting and depressed ankles and May be impaired Pin prick and joint position with high steppage gate and positive Romberg's Test

**Cerebellar signs** Impaired heel shin testing

**Lower limbs** in Friedreich's ataxia

**Bilateral Pes Cavus**

**Distal wasting and Pyramidal weakness with evidence of**

- Hypotonia/Normal tone
- Depressed ankles
- Upgoing plantars
- Impaired heel shin test
- Impaired Pin Prick sensations in the Stocking distribution (May not be evident)
- Impaired joint position sensation and Vibration

**Gait**

- Broad based
- Ataxic on both sides
- Romberg's test is positive

**Upper limbs**

- Features of bilateral cerebellar involvement

**Eyes**

- Nystagmus

**Speech**

- Slurred

**I would like to complete my examination by:**

Taking detailed history

By complete neurological examination

Fundoscopy (DM,OA)

Examination of hearing,Heart

Dip the urine for Glycosuria

### Differential diagnosis:

**Depressed ankles and upgoing plantars**

#### P-MAST

1. Peripheral Neuropathy + Cervical myelopathy or Bilateral stroke
2. MND
3. FA
4. SACD
5. Taboparesis

### Investigations:

- Blood tests e.g Fasting blood sugar and Hb A<sub>1c</sub>
- NCS
- Visual evoked potentials
- ECG and Echocardiography
- Imaging of Brain and spinal cord
- Genetic Testing

### Treatment:

#### General measures:

#### Patient's education

#### MDT care package involving

- Physiotherapist
- Neurologist
- Orthopedic surgeon
- Occupational health physician
- Speech and Language therapist

**Medical treatment:**

**Symptomatic treatment**

**Screening and treatment of DM**

**Screening and treatment of Heart problem**

**Surgical:**

**Corrections if required**

## 9. Hemiparesis (Stroke most likely)

Common in PACES exam

### Commands:

- Examine the Upper Limbs of this gentleman
- Examine the Lower Limbs of this gentleman
- Examine the gait and proceed

Examination scheme as advised and done in routine, especially :

- Gait
- Motor system
- Coordination

Associated e.g

- Pulse
- Listening to carotids and Heart

I would like to complete my examination by

- Taking detailed History
- Complete the neurological examination including visual field testing
- Do the Fundoscopy
- Check the Blood pressure and
- Dip the urine for glycosuria

## Recording of Lower Limb examination

Well,

I have examined this gentleman,

He has Upper Motor Neuron type weakness in the Right Lower Limb as evidenced by:

- Extensor Posturing
- Hypertonia
- Hyper-reflexia
- Upgoing plantar
- With normal coordination (If applicable)

And

- Circumducting gait (Hemiplegic)

So based on this, my most likely diagnosis is Stroke

DD:

- SOL brain like Tumor,abscess,tuberculoma etc
- Demyelination (MS)
- Todd's Paresis
- Functional

### Investigations:

#### General:

- CBC,ESR,CRP
- Fasting blood sugar and Hb A<sub>1c</sub>
- Fasting blood sugar
- Fasting lipid profile

Specific depending upon the suspected cause like looking for cause of stroke in young

- ECG
- Echocardiography and others etc
- Imaging
- CT brain
- MRI,MRV,MRA brain etc

Four Vessels Neck Doppler to document the narrowing

### Treatment:

#### General:

As stated previously in other cases

#### Medical:

Depends upon the cause

## 10.Motor Neuron Disease

**A progressive disorder which is characterized by neuronal loss at all levels of the motor system**

**Characterized by both upper and lower motor neuron features in the same muscle group**

**Sensations** are normal

**There may be evidence of Bulbar/Pseudo-bulbar involvement**

**Bulbar palsy** means LMNs of 9<sup>th</sup>,10<sup>th</sup>,11<sup>th</sup> and 12<sup>th</sup> may be involved

**Other cranial nerves** may also be affected like 5<sup>th</sup> and 7<sup>th</sup> cranial nerves motor function may be affected

**Oculomotor muscles** are spared in MND

**Speech** may be slurred or nasal in quality

**Pseudo-bulbar** involvement may cause following features:

- **Emotional Lability** with spontaneous Laughing and crying
- **Small stiff tongue**
- **Pronounce gag reflex**
- **Brisk jaw reflex**
- **Speech may be slow and thick like 'hot potato speech'**

### Types of MND

1. **Amyotrophic Lateral sclerosis (ALS)** May have both upper and LMN features
2. **Primary Lateral Sclerosis (PLS)** Initially may have UMN signs only
3. **Progressive Muscular Atrophy (PMA)** May have LMN signs initially
4. **Progressive Bulbar Palsy (PBP)** May present initially with LMN (**Bulbar**) or UMN (**Pseudo-bulbar**) signs

### Differential Diagnosis:

1. **Chronic inflammatory demyelinating polyneuropathy (CIDP)**

2. **Myasthenia Gravis:**

- Autoimmune neuromuscular disorder, Fluctuating symptoms of skeletal muscles weakness ,fatigueability and extraocular muscles may be involved

3. **Poliomyelitis Syndrome**

4. **Dual pathology like Cervical Myelopathy and Peripheral Neuropathy**

5. **Syringomyelia/Syringo-bulbia**

- May have LMN signs with disassociated sensory loss at the level of the lesion and UMN signs below the lesion

6. **Inclusion Body Myositis**

- Which is an inflammatory condition may develop in the elderly and may present with distal and proximal muscle weakness without sensory involvement and no UMN features .EMG and Muscle biopsy may be helpful

7. **Spinal Muscular atrophy (SMA)**

- May diagnosed usually in infancy and rarely in early adulthood

### Investigations:

Diagnosis is clinical

Investigations like MRI,CSF analysis ,NCS,EMG and Muscle biopsy are done to rule out certain other possibilities

**Treatment:**

**General Measures:** Patient's and family education and explanation of the condition

MDT care package including

- Neurologist
- Physiotherapist
- Dietician
- Occupational therapist
- Speech and language specialist etc

Build up nutrition

Vaccinations etc

**Medical treatment:**

The only disease modifying treatment is **Riluzole** which can **prolong life for 3-4 months** which should be offered to all.

**Treatment of complications:**

**Depression:**

- Amitriptyline or SSRI

**Drooling:**

- Anti-cholinergic drugs, TCA, Radiotherapy to the parotids and home suction therapy etc

**Communication problems:**

- Speech and Language therapist

**Pathological laughing or crying:**

- Amitriptyline and Lithium etc

**Dysphagia:**

- Dietary advise and PEG insertion

**Respiratory problems:**

- Cough augmentation devices, Physiotherapy, Suction and Non-invasive respiratory support

**Fasciculations and muscle cramps:**

- Magnesium, Vitamin E, Diazepam, quinine, carbamazepine and phenytoin etc

**Spasticity:** Physiotherapy and muscle relaxants like Baclofen and Dantrolene etc

## 11. Multiple sclerosis

An inflammatory demyelinating disorder of the CNS.

Lesions separated in space and time i.e at least there must be 2 distinct episodes of neurological dysfunction to support a diagnosis of MS (So will be possible on history to support this on clinical grounds)

Mixtures of neurological symptoms and signs that do not fit into a single diagnosis favors the MS

Usually young female patient or middle aged lady

Signs could be unilateral or bilateral

Common sites involved are

- Optic nerve
- Brainstem
- Cerebellum
- Spinal cord with Cortico-spinal and Dorsal column most likely and rarely spino-thalamic tracts.

**Commands:**

- Examine the eyes as this patient has presented with painful blurriness.
- Examine the speech of speech and proceed accordingly.
- This patient has presented with balance problem, examine her neurological system.
- This patient has walking difficulty, examine her.
- Examine the upper limbs of this lady.

**Possible findings on eye examination:**

- Decreased vision and decreased color vision with optic atrophy on fundoscopy
- Internuclear ophthalmoplegia (Usually bilateral)
- Relative afferent pupillary defect (RAPD)
- Ipsilateral or bilateral Nystagmus because of Cerebellar involvement with or without INO

### Differential diagnosis:

With Spastic paraparesis

#### C-MAST

- Cervical myelopathy
- Friedrich ataxia
- Subacute combined degeneration
- Taboparesis

And

- Ischemic stroke (If unilateral involvement)

### Types:

Relapsing-remitting (80-85 %)

- Most common
- Acute and short exacerbation
- Followed by partial or complete recovery

Secondary progressive

- Most patients with relapsing-remitting may evolve into this form without acute exacerbations

Primary progressive

- Progressive from onset

Relapsing progressive

- Progressive with marked deteriorations

### **Investigations of Multiple sclerosis:**

**Diagnosis is usually clinical but can be supported with the help of imaging**

**Blood tests are done to rule out other causes**

- CBC,ESR,CRP
- Thyroid profile
- Vitamin B -12 levels

**T2 weighted MRI**

**MRI of the cervical cord** to differentiae from cervical spondylsos

**Visual Evoked Potentials (VEP)** shows asymmetrical and prolonged conduction

**Lumbar puncture with CSF analysis** IgG Oligoclonal bands In greater concentrations

**Anti-NMO antibodies** to differentiate form Devic's desease

## Management:

The aim is to prevent disability and improvement in quality of life

### General measures:

- Patient's education and explanation of the condition
- Education of the family and carers
- MDT care package involving different professionals
- Vaccinations
- Support organizations like Multiple Sclerosis Society

## Medical treatment:

### 1.Treatment of acute attack:

- **Steroids:** I/V or Oral Methylprednisolone for 3-5 days
- **IV Immunoglobulins** as second line option

### 2.Secondary prevention:

#### Relapsing remitting and relapsing progressive

- Interferon Beta or Glatiramer
- Natalizumab

#### Secondary progressive

- Mitoxantrone

#### Primary progressive

Currently not recommended treatment however the above options can be used

Other immunosuppressive agents can be tried like

- Azathioprine
- Methotrexate
- Cyclophosphamide

### 3.Treatment of complications:

- Fatigue
- Cognitive symptoms
- Mood changes
- Osteoporosis
- Bowel dysfunction
- Urinary symptoms
- Gait problems

## 12.CP Angle Syndrome

### General

- Facial asymmetry
- Front of ear
- Back of ear
- Inside ear for vesicles
- Inside mouth for vesicles

### Then

- Facial nerve examination

### Followed by

- V, VI, VIII Nerves
- Stroke
- Cerebellum

### Other Features to look for when facial nerve involvement

- Scar
- Parotid swelling
- Vesicles
- Back of neck for scar
- Weakness of arms

### **Other topics to study for PACES**

- 1. Visual field defects**
- 2. Wasting of small muscles of hands**
- 3. Horner's syndrome**
- 4. Speech like Dysphasia**
- 5. Old poliomyelitis**
- 6. Muscular dystrophy**
- 7. Brainstem syndrome**
- 8. Radial, Median and ulnar nerves**
- 9. Involuntary movements**









# **Respiratory System**

## Respiratory System

### General

- Cachectic
- Cushingoid
- Breathless
- Others

### Hands

- Clubbing
- Cyanosis
- Nicotine staining
- Tremors
- Wasting
- Others- e.g Ulcers, scars, pitting of nails , sclerodactyly etc

### Pulse

- Good volume
- Regular or irregular

### Skin

- Purpura
- Bruising
- Striae
- Smooth & shiny skin at the back of hands

### JVP

- Not raised (raised)

## Face

### Eyes

- Pallor
- Jaundice

### Mouth

- Telangiectasia
- Candidiasis
- Cyanosis

### Others

- Thinning of nose
- Reduce opening of mouth
- Puckering of skin around the mouth

## Lymph nodes

## Chest Inspection:

- Hyper inflated
- Depressed
- Throacotomy scar (right / left)

## Trachea

- Central
- Deviated

## In the right /left (upper, middle, lower) part of the chest

1. Chest expansion is.....
2. Precaution note is.....
3. Breath sounds are .....
4. Vocal resonance is .....

## Peripheral Edema

I would like to complete my examination by

- Taking detailed history
- Check the oxygen saturation and Peak flow rate
- Look into the sputum
- And check the chart for vitals

## **Recordings of Respiratory system**

## 1. Interstitial Lung Disease & Steroid Therapy

I would like to complete my examination by:

Taking detailed history

Check the observation chart

Check the Oxygen saturation at bed site

Do the Peak flow metry and

Look into the sputum mug

I have examined this Lady:

She has evidence of:

Cushingoid appearance and

Breathless at rest (with oxygen therapy)

There is evidence of Clubbing and Peripheral cyanosis

Multiple Striae

JVP is Not raised

Chest is of normal shape

Trachea is central in position

In the lower part of chest on both sides

1. Chest expansion is reduced
2. Percussion note is dull (or normal)
3. There are end inspiratory crackles that do not change on coughing
4. Vocal resonance is reduced (or normal)

## 2. Interstitial Lung diseases + Scleroderma + Pulmonary HTN + Steroid Therapy

I would like to complete my examination by:

Taking detailed history

Check the observation chart

Check the Oxygen saturation at bed site

Do the Peak flow metry and

Look into the sputum mug

I have examined this Lady:

She has:

Cushingoid appearance and breathless at rest (and on oxygen therapy)

In the Hands there is evidence of :

- Clubbing
- Peripheral synopsis
- Ulcers ,Pitting, Scars, Infarcts of finger tips.
- Sclerodactyly
- Smooth and shinny skin at the back of hands

She has Purpura and Striae

JVP is raised / not raised

There is evidence of:

Thinning of nose

Puckering of skin around the mouth

Reduced opening of mouth

**Chest is:**

**Of Normal shape and Trachea is central in position**

**In the lower part of the chest on the both side:**

- **Chest Expansion is reduced**
- **Precession Note is dull (Normal)**
- **Breath sound are reduced and there are inspiratory crackles that don't change on coughing**
- **Vocal Resonance is reduced (Normal)**

**There is also evidence of left Parasternal heave and the pulmonary component of the 2<sup>nd</sup> heart sound is also loud.**

**Peripheral edema is also present**

## CAUSES OF PULMONARY FIBROSIS:

**Idiopathic pulmonary fibrosis** is the most common cause (Idiopathic interstitial pneumonia)

Others are associated with different conditions like:

- **Rheumatological or connective tissue disorders** like RA,SLE,scleroderma,Ankylosing Spondylitis,dermatomyositis etc
- **Vasculitides** like PAN,Church Strauss Syndrome etc
- **Hypersensitivity pneumonitis** like Bird Fancier's lung,wood worker's lung etc
- **Occupational pneumoconiosis** Coal worker's lung, asbestosis and metal dust like berellyosis etc
- **Radiation or radiotherapy**
- **Smoking**
- **Tuberculosis** may cause asymmetrical fibrosis
- **Genetic form**
- **Medications** Methotrexate,Amiodarone, Bleomycine,gold,Sulfasalazine and cyclophosphamide etc

## Differential diagnosis:

- **Bronchiectasis**
- **Pulmonary edema**
- **Non-cardiogenic pulmonary edema**

**General investigations:**

**CBC** to check for polycythemia and WBCs response of active infection

**Inflammatory markers** like ESR,CRP

**Specific investigations:** Depends upon suspected underlying cause:

- Rheumatoid factor, anti-cyclic citrulline peptide antibodies (ACCP antibodies)
- ANA, ASMA, Anti-ds DNA antibodies
- c-ANCA, p-ANCA
- CPK
- Anti-Scl 70 antibodies
- Serum ACE level
- Immunoglobulins
- Precipitins etc

**Others:**

**6 Minutes walk test** to check functional status and desaturation

**ABGs** to document Type 1 respiratory failure

**Pulmonary function tests** to document if there is Restrictive pattern and to measure lung volumes

### Imaging:

#### CXR-PA view to document:

- Reduced Lung volumes
- Reticulonodular shadowing and Honey comb appearance in advanced cases
- Other findings like Lymphadenopathy and calcified plaques etc

**HRCT:** To document pattern and distribution like

Ground glass appearance suggests good prognosis and responds well to steroids and immunosuppressive therapy

#### Bronchoalveolar lavage (BAL):

- If Lymphocyte predominant, it means good prognosis
- Other cells like Eosinophils and Malignant cell can be sought as well

**Lung biopsy:** Sometimes needed to reach a specific cause

- Trans-bronchial
- Video-assisted thoracic surgery (VATS)
- Open biopsy procedure

**Echocardiography and Right heart Catheterization** sometimes required if there is suspicion of Cor-pulmonale

**Treatment:**

**General Measures:**

- Patient's education and explanation of the condition
- MDT care package
- Smoking cessation and avoidance of the triggers at job and at home
- Discontinuation of toxic medicines if taking
- Build up nutrition
- LTOT if required
- Vaccinations like Influenza and Pneumococcal
- Pulmonary rehabilitation exercises

**Medical treatment:**

Treatment of underlying condition if possible with specific medicine if applicable

**Others include following:**

- Corticosteroids
- Immunosuppressive therapy like Azathioprine
- Antifibrotic agents like Colchicine and D-Penicillamine etc

**Treatment of complications like:**

- Infections with prompt use of Antibiotics
- Treatment of Cor-pulmonale etc

**Surgical measures:**

With sever disease and age less than 65 years, Single OR Double lung transplantation can be offered

### 3. Chronic Obstructive Pulmonary Disease (COPD)

I would like to complete my examination by:

Taking detailed history

Check the observation chart

Check the Oxygen saturation at bed site

Do the Peak Flowmetry and

Look into the sputum mug

I have examined this Gentleman/Lady:

He/She is:

Cachectic ,has Cushingoid appearance and breathless at rest

In hands there is evidence of:

Nicotine staining

Tremors

Asterixis

Pulse is regular

JVP is not raised

He/She also has evidence of: Jaundice and Cyanosis

Chest is Hyper inflated

Trachea is Central in position

In whole of the chest on both sides

- Chest expansion is reduced
- Percussion note is hyper-resonant
- Breath sounds are reduced and there is evidence of prolonged expiration and expiratory wheezing
- Vocal resonance is also reduced.

### Differential Diagnosis:

- Asthma
- Bronchiolitis Obliterans

### Investigations:

#### General investigations:

- **CBC** to check for WBCs response and Hb to rule out secondary polycythemia
- **Inflammatory markers** like ESR and CRP for suspected underlying infection
- **LFTs** if suspicion of Cor-pulmonale or Alfa-1 anti-trypsin deficiency
- **Serum electrolytes** especially Potassium level

#### Specific:

**Sputum analysis** including culture if required

**Alfa 1 antitrypsin assay**

#### ABGs:

- To check for Type 2 respiratory failure and Respiratory acidosis if exacerbation
- Also to assess for LTOT

**Lung function tests** to check if there is Obstructive pattern and lung volume assessments

And **GOLD stage** can be used to measure airflow obstruction

**CXR-PA View** to look for

- Hyper-inflated lungs
- Flattening of diaphragms
- Tubular heart
- Bullae etc

**HRCT:** Most sensitive for diagnosing Emphysema

**Other tests include:**

**ECG** to check for right ventricular hypertrophy and arrhythmias like MAT, Wandering pacemaker and atrial fibrillation etc (Due to deoxygenation)

**Echocardiography and Right heart catheterization study to for suspected Cor-pulmonale**

**Treatment:**

**General measures:**

Pulmonary rehabilitation with the help of MDT involving

- Patient's education and explanation of the condition
- Build up nutrition
- Psychological support
- Physical training

Smoking cessation

Vaccinations like Influenza and Pneumococcal vaccines

**Medical treatment:**

**Bronchodilators** like Beta-agonists and Anti-cholinergics

**Anti-inflammatory therapy** in the form of Oral/IV Corticosteroids in acute exacerbations and long term inhaled corticosteroids as maintenance therapy

**Criteria for LTOT:**

Patient should have stopped smoking

Supplementary oxygen therapy can be used for more than 15 hours and sometimes more than 20 hours

LTOT can be considered in the following patients group

- Cyanosis
- Raised JVP
- Peripheral edema
- Oxygen saturation < 92 % in air when stable
- Polycythemia
- FEV<sub>1</sub> < 30 % predicted

Indications for LTOT in patients in which 2 ABGs are measured 2 weeks apart with following readings:

**PaO<sub>2</sub> < 7.3 K Pa**

OR

**PaO<sub>2</sub> < 8 K Pa with one of the following:**

- Secondary polycythemia
- Pulmonary HTN
- Peripheral edema
- Nocturnal Hypoxemia

#### 4. Pleural Effusion (right sided)

I would like to complete my examination by:

Taking detailed history

Check the observation chart

Check the Oxygen saturation at bed site

Do the Peak flow metry and

Look into the sputum mug

I have examined this Gentleman/Lady:

He/She is:

Cachectic and has

Nicotine Staining

Clubbing and wasting of Small muscles of Right hand

Pallor, with evidence of Horner Syndrome

Left cervical Lymph Nodes are palpable

Right lower part of the chest is bulging

Trachea is Central in positoin

(Deviated towards other side only if massive effusion or same side if there is collapse on the same side as well)

In the right lower part of the chest:

- Chest expansion is reduced
- Percussion note is stony dull
- Breath sound are reduced
- Vocal resonance is also reduced

There is no Edema

**Differential diagnosis:**

**Causes:**

**Exudative:** Protein content > 3gm/dl

- **Infections** like Pneumonia,TB etc
- **Inflammation** like RA,SLE etc
- **Neoplastic** like CA Lung,Metastasis,Mesothelioma and Meig's syndrome etc
- **Pulmonary infarction** as may occur in Pulmonary embolism
- **Drugs** like methotrexate and nitrofurantoin etc

**Transudative:** Protein content < 3 gm/dl

### Investigations:

#### General investigations:

- CBC to look for anemia and WBCs response
- Inflammatory markers like ESR,CRP
- LFTs especially if atypical pneumonia suspected as a possible cause
- Serum electrolytes like Hyponatremia may occur in atypical pneumonias

#### Specific investigations:

Depends upon underlying suspected cause, however following can be used:

- Serum LDH to be used in Light's Criteria
- Serum albumin and Lipid profile
- Serum amylase
- TFTs
- Rheumatoid factor
- Autoimmune profile (ANA, ASMA, ANCA)
- Sputum analysis including culture and sensitivity

**Pleural tap** For Cell count, biochemistry and Culture and Sensitivity and other possible investigations depending upon the cause

#### ABGs

#### Imaging:

CXR-PA view

CT scan of the chest if suspected malignancy

Other possible investigations include:

- Lung biopsy
- Bronchoscopy
- Echocardiography
- Mammography
- CT abdomen
- PET
- Bone scan

Light's criteria for Exudative effusion:

- Pleural fluid Protein:Serum protein > 0.5
- Pleural fluid LDH:Serum LDH > 0.6
- Pleural fluid LDH > 2/3 of the upper Limit of normal serum value

**Treatment:** Depends upon the underlying cause

**General measures:**

- Patient's education
- MDT care package
- Build up nutrition
- Smoking cessation if applicable
- Salt and fluid restriction if required

**Specific Treatment:** Depends upon underlying cause e.g for CA lung following measures depending upon the stage of the tumor

**Surgical resection:** For Non-SCLC but not suitable for Small Cell Lung Cancer

**Chemotherapy:** Cisplatin based regimen can be used in SCLC and can be as an adjunct in case of Non-SCLC

**Radiotherapy:** As an adjunctive or alternative treatment for both SCLC and NSCL

**Therapeutic Pleural tap:** For symptomatic relief

**Palliative Treatment:** Depends upon the patient needs

## 5. Bronchiectasis

I would like to complete my examination by:

Taking detailed history

Check the observation chart

Check the Oxygen saturation at bed site

Do the Peak flow metry and

Look into the sputum mug

I have examined this Gentleman/Lady:

He/She is:

Cachectic and has Clubbing

Trachea is central in position

In the right lower part of the chest

- Chest expansion is reduced
- Percussion note is Resonant/Dull
- There are inspiratory crackles that change on coughing
- Vocal resonance is Reduced/Normal

There is no Peripheral Edema

**Causes:**

**Childhood infections**

- TB
- Measles

**Chronic adult infections (Especially in immunodeficiency states like HIV)**

- TB
- Pneumonia

**Congenital defects**

- Yellow nail syndrome

**Ciliary dyskinesias**

- Karttgener's syndrome
- Cystic fibrosis

**Autoimmune diseases**

- RA
- IBD

**Allergic bronchopulmonary aspergillosis**

**Bronchial obstruction:**

- Foreign body
- Lymphadenopathy

**Idiopathic**

**Causes of clubbing with Crackles in the chest**

- Bronchiectasis
- ILD
- Lung cancer
- Lung abscess

### Investigations:

#### General:

**CBC** to look for anemia and WBCs response

**Inflammatory markers** like ESR,CRP

**Specific:** Depends upon suspected cause

- Sputum analysis especially culture and sensitivity,AFB staining
- Serum ACE-level
- HIV serology

#### Immunology:

- Immunoglobulins
- RA factor , ANA,ASMA,Anti – ds DNA etc
- Aspergillus presiptians,Skin prick testing

**Saccharine ciliary motility test**

**Genetic Screening**

**PFTs:** To document Obstructive or restrictive pattern

#### Imaging

**CXR-PA View** to Look for Ring shadows and Tram lines

**HRCT** to look for Signet ring sign

**Bronchoscopy and biopsy**

### Treatment of Bronchiectasis:

#### General measures:

Oxygen therapy may be required if low oxygen saturation on rest

#### Pulmonary Rehabilitation

- Patient's education and explanation of the condition
- MDT care to deal different problems
- Build up nutrition
- Psychological and physical support

#### Smoking cessation

#### Avoidance of precipitans in ABPA

#### Postural drainage and Physiotherapy

#### Medical treatment:

**Bronchodilators** Anti-cholinergics if obstructive pattern

**Treatment of complications** Prompt control of infections with antibiotics

**Surgical measures:** Surgical resection for localized disease not controlled by medical treatment

**Bronchial artery Embolization** If massive hemoptysis

#### Related topics to study

- Yellow nail syndrome
- ABPA
- Kartagener's syndrome

## 6. Right lower Lobectomy

I would like to complete my examination by:

Taking detailed history

Check the observation chart

Check the Oxygen saturation at bed site

Do the Peak flow metry and

Look into the sputum mug

I have examined this Gentleman/Lady:

He/She has evidence of:

Nicotine staining

Clubbing, wasting

Pallor

Left cervical Lymph adenopathy

There is evidence of Thoracotomy scar on the right side

Trachea is central in position

In the Right Lower part of the chest:

- Chest expansion is reduced
- Percussion note is dull
- Breath sounds are Absent/Reduced
- Vocal resonance is also reduced

## Other respiratory cases

1: Rheumatoid Lung

2: Lobectomy

Right

Upper

Lower

Left

Upper

Lower

3: Penemonectomy

4: Consolidation

5: Effusion + Collapse

6: Malignancy (With evidence of Tattoo marks for radiotherapy)

7: Old Tuberculosis

Apical Fibrosis

Thoracoplasty

Pneumonectomy

Lobectomy

Plombage Therapy

Phrenic nerve crush

Recurrent Pneumothoraxis

8: Lung collapse

9. Lung Transplant (Single or Double)









# Abdomen

## Possible Abdominal Cases

Liver / Spleen	Ascites	Renal	Scars	Mass
CLD	CLD	APKD	Renal Transplant	Renal single or bilateral
Isolated Splenomegaly	CCF	Renal Transplant	Pancreatic Transplant	APKD Pancreatic
Hepato-splenomegaly	Localized e.g <ul style="list-style-type: none"> <li>• Malignancy</li> <li>• Tuberculosis</li> </ul>	Hemodialysis	Liver transplant and Donors scar	Epigastric
Isolated Hepatomegaly	Others	Peritoneal Dialysis	Splenectomy	Right Iliac Fossa
Liver Transplant	<ul style="list-style-type: none"> <li>• Cor-pulmonale</li> <li>• Budd chiari syndrome</li> <li>• Nephrotic syndrome</li> <li>• Serositis e.g RA,SLE etc</li> </ul>	Nephrotic Syndrome Nephrostomy Insulin Pump	Lapotomy Nephrectomy Appendicetomy Pfannenstein Multiple scars of Crohn's disease Multiple scars of Crohn's disease	

## Stigmata of CLD

- **Leuonychia**
- **Clubbing**
- **Palmar Erythema**
- **Duputyren's Contracture (Alcoholic)**
- **Flapping Tremors**
- **Jaundice**
- **Cyanosis**
- **Spider Angioma**
- **Gynaecomastia (Male)**
- **Decreased Body Hair**
- **Engorged veins, Caput Medusae**
- **Purpura & Ecchymosis**
- **Testicular atrophy**

## Causes of CLD

Causes	Clues
<b>Alcohol</b>	Duputren's contracture  Bilateral parotid swelling  Multiple spider nevi
<b>Viral</b>	Piercing and Tatoos etc
<b>Autoimmune</b> <ul style="list-style-type: none"> <li>• PBC</li> <li>• PSC</li> <li>• Autoimmune Hepatitis</li> </ul>	Usually Female  Other autoimmune clues like  Alopecia,Vitiligo,Thyroid problem,Type 1 DM etc
<b>Metabolic</b> <ul style="list-style-type: none"> <li>• Willson's Disease</li> <li>• Alpha 1 Anti-Trypsin Deficiency</li> <li>• Hemochromatosis</li> <li>• NAFLD</li> </ul>	CLD in young plus Extrapyramidal features in Wilson's Disease  Young with COPD and CLD in alpha 1 anti trypsin deficiency  May be knee replacement in Hemochromatosis
<b>Vascular</b> <ul style="list-style-type: none"> <li>• BCS</li> <li>• CCF</li> </ul>	History of DVT, tender Hepatomegaly,ascites in BCS  Features of CCF
<b>Drugs</b> <ul style="list-style-type: none"> <li>• Methotrexate</li> <li>• Amiodarone</li> <li>• Phenytoin</li> <li>• INH</li> </ul>	History of medicines intake

Hepatomegaly	Hepato-splenomegaly	Isolated Splenomegaly
<b>CLD</b> <ul style="list-style-type: none"> <li>• NAFLD</li> <li>• Alcoholic Liver Disease</li> <li>• Hemochromatosis</li> <li>• PBC</li> <li>• HCC</li> </ul>	<b>CLD</b>	<b>CLD</b>  If no features of CLD consider non – cirrhotic portal hypertension ( <b>NCPHTN</b> ) with ascites or upper GI bleed and isolated splenomegaly
<b>Metastatic</b>	<b>Hematological Conditions</b> <ul style="list-style-type: none"> <li>❖ Myeloproliferative</li> <li>❖ Lymphoproliferative</li> <li>❖ Hereditary Hemolytic anemias like Thalessemia and Hereditary Spherocytosis</li> </ul>	<b>Hematological conditions</b> <ul style="list-style-type: none"> <li>• Myeloproliferative</li> <li>• Lymphoproliferative</li> <li>• Hereditary Hemolytic anemias like Thalessemia and Hereditary spherocytosis</li> </ul>
<b>Vascular</b> <ul style="list-style-type: none"> <li>• CCF</li> <li>• BCS</li> </ul>		
<b>Infective</b> <ul style="list-style-type: none"> <li>• Viral</li> <li>• Abscess</li> <li>• Hydatid cyst</li> </ul>	<b>Infective</b> <ul style="list-style-type: none"> <li>• Viral e.g HIV,EBV,CMV</li> <li>• Bacterial e.g Disseminated TB,Typhoid,Brucellosis etc</li> <li>• Parasitic e.g Malaria,leishmaniasis,schistosmiasis</li> </ul>	<b>Infective e.g</b> <ul style="list-style-type: none"> <li>• Infective endocarditis</li> <li>• Malaria</li> <li>• Kala azar</li> </ul>
<b>.Infiltrative</b> <ul style="list-style-type: none"> <li>• Amyloidosis</li> <li>• Sarcoidosis</li> </ul>	<b>Infiltrative</b> <ul style="list-style-type: none"> <li>• Amyloidosis</li> <li>• Sarcoidosis</li> </ul>	<b>Inflammatory e.g</b> <ul style="list-style-type: none"> <li>• SLE</li> <li>• RA</li> </ul>
<b>Polycystic Liver</b>	<b>Storage Diseases e.g Gaucher's Disease,Glycogen storage disease</b>	<b>Storage diseases</b>
<b>Riedel's Lobe</b>	<b>others</b>	<b>Pernicious anemia</b>

## Chronic Liver Disease

**Investigations:**

**General investigations:**

**CBC** to look for Anemia and Thrombocytopenia

**LFTs** to establish the severity e.g

- Raised bilirubin
- Transaminases
- Synthetic function by determining Low albumin and Raised PT,INR

**RFTs** to look for Hepato-renal syndrome with other criteria

**Serum electrolytes**

**Specific investigation:**

To establish the cause:

**Viral serology like**

- HBs Ag, HCV antibodies

**Autoimmune profile like**

- ANA,ASMA,Anti-LKM 1,serum Immunoglobulins
- Anti-Mitochondrial antibodies

**Metabolic Profile like**

- Serum Ferritin
- Serum Ceruloplasmin
- Alfa 1 anti-trypsin level

**Others according to the cause**

**Imaging:** e.g

**USG abdomen** to confirm the findings e.g

- Coarse echotexture of the liver
- Mass
- Ascites and Splenomegaly

**CT scan** of to rule out hepatoma

**Doppler Scan** for vascular patency

**Others like:**

**Diagnostic ascetic tap** for

- Cell count with differentials
- Biochemistry
- Culture and sensitivity

**Alfa feto protein** if suspicion of mass

**Liver biopsy** for staging and determination of the cause if required

**Oesophago-gastro-duodenoscopy (OGD)** for surveillance of Varices

**Other non-invasive tests for Cirrhosis**

- Transient Elastography
- Acoustic radiation force Elastography (ARFI)
- Magnetic resonance Elastography (MRE)

**Treatment:**

**General measures:**

- Patient's education and explanation of the condition
- MDT care package
- Build up nutrition
- Vaccination especially against HAV, HBV, Influenza and pneumococcus
- Salt restriction (If ascites)
- Daily weight measurement and abdominal girth if patient has ascites and on diuretics

**Specific therapy:**

Depends upon underlying cause:

- Alcohol reduction and abstinence
- Antiviral therapy against HBV and HCV
- Steroids and immunosuppressive for autoimmune diseases
- Specific therapy for metabolic causes accordingly

**Treatment of complications:**

**Hepatic Encephalopathy:**

- Find the triggers and fix it
- Antibiotics
- Gut purgatives like Lactulose

**Upper GI bleeding:**

- EGD with banding and sclerotherapy
- Beta-blockers

**Ascites:**

- Salt restriction
- Diuretics
- Therapeutic paracentesis

**SBP:**

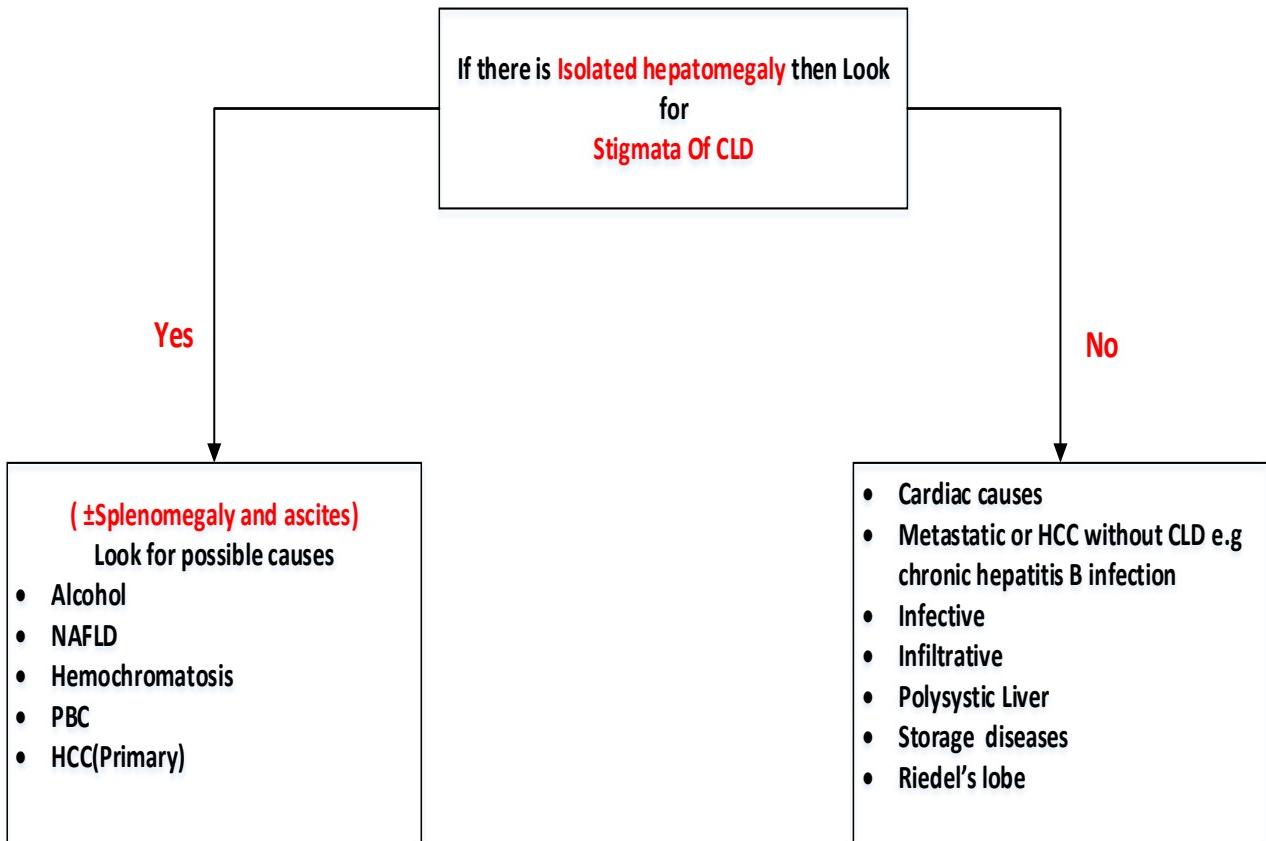
- Anitbiotics
- Albumin infusion

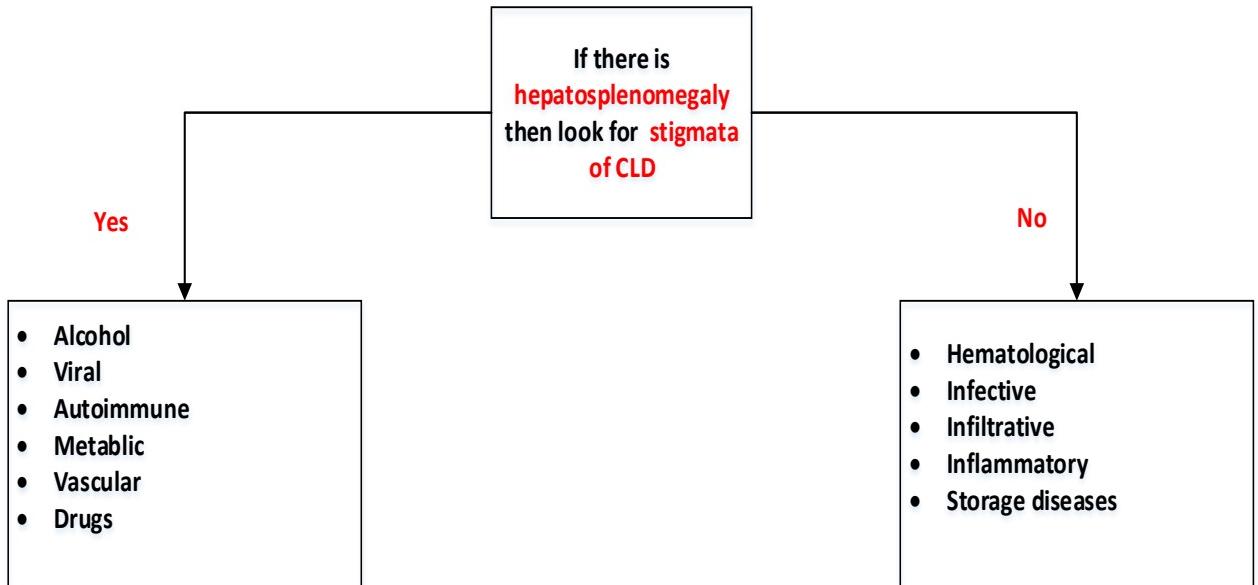
**Hepatorenal syndrome:**

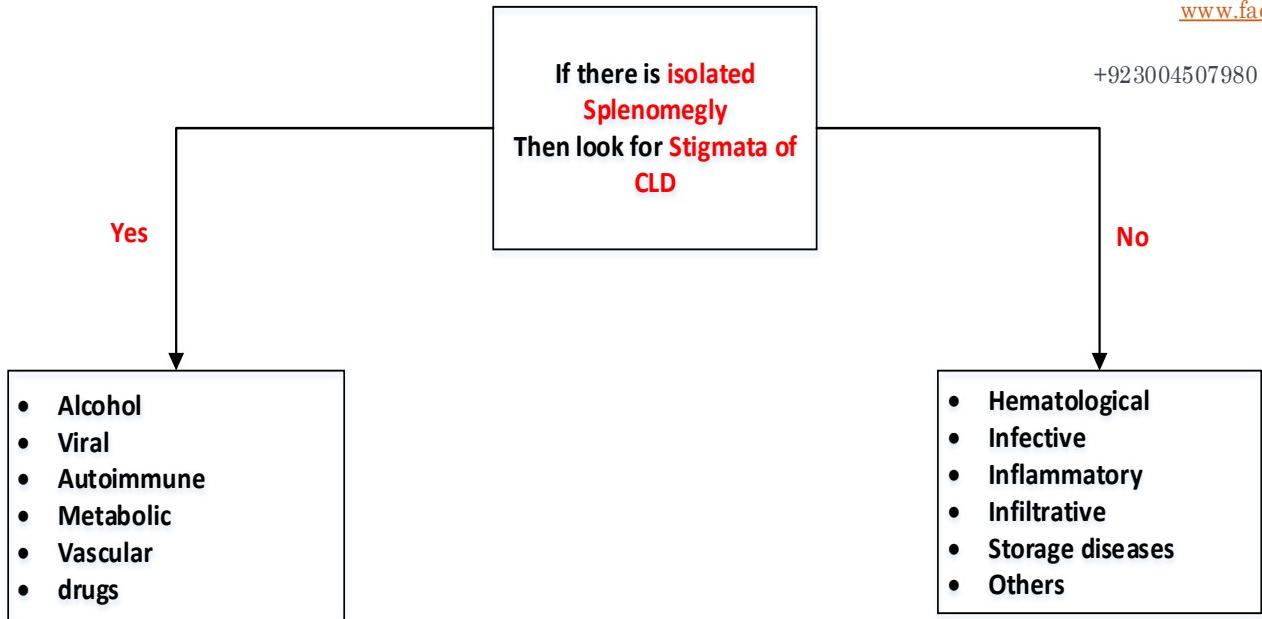
- Albumin infusion
- Vasoconstrictives like Terlipressin

**Hepatoma :**

- **Surgical resection**
- **Liver Transplantation**
- **Percutaneous ethanol injection/RFA**
- **Transarterial chemoembolization (TACE)**
- **Chemotherapy like Sorafenib**
- **Palliative**







### Causes of Massive Splenomegaly

#### 1. Myeloproliferative disorders

- CML
- Myelofibrosis

#### 2. Parasitic infections like

- Malaria
- Kala azar

## **CLD with Splenomegaly OR Hepatosplenomegaly OR Hepatomegaly with Or without Ascites**

**I would like to complete my examination by:**

Taking detailed history

Check the observation charts

Examine the external genitalia and hernial orifices

And do the Digital rectal examination (If melena etc)

**I have examined this Gentleman/Lady**

**He/She is:**

Cachectic

**In the Hands there is evidence of:**

Clubbing

Leuconychia

Palmar erythema

Duputyn's contractures

Flapping Tremors (If Encephalopathy)

**In the arms, there is evidence of:**

Scratch marks and bruising

**On further examination, there is evidence of:**

Pallor

Jaundice

Multiple spider nevi in the chest

Bilateral gynaecomastia

Decreased Body hair

**On examination of the abdomen:**

**It is distended with evidence of prominent veins, white striae and umbilical hernia**

**But soft and non-tender**

**There is evidence of splenomegaly (Hepatosplenomegaly/Hepatomegaly)**

**with splenic edge being palpable 3 fingers breadth below the left costal margin which is firm, smooth and non-tender and moves with respiration.**

**There is clinically detectable ascites as evidenced by shifting dullness**

**Bowel sounds are audible**

**However, there is No evidence of:**

**Lymphadenopathy**

**And**

**Peripheral edema is not present**

**So based on this my most likely diagnosis is Cirrhosis with Portal Hypertension**

**Cause depends upon underling clues present:**

**Causes include:**

1. Alcohol
2. Viral
3. Autoimmune
4. Metabolic
5. Vascular
6. Drugs and toxins
7. others

## Liver Transplant

**Additional Features include following:**

- **Cushingoid appearance**
- **Thinning of skin**
- **Pin prick marks in the fingers**
- **Thinning of skin**
- **Busing**
- **Striae**
- **Supraclavicular and inter-scapular fat pads**
- **Cushingoid face with excessive hair and acne**
- **Mercedes Benz (Roof top scar which is non-tender)**
- **Tremors in the hands**
- **Gum hypertrophy**
- **Papillomas and skin warts**
- **others**

## Renal Transplant

I would like to complete my examination by:

Taking detailed history

Check the observation chart and blood pressure

Dip the urine for hematuria and proteinuria

I have examined this Gentleman/Lady:

He/She has:

Cushingoid appearance

With evidence of:

Pin-prick marks

Tremors

AV fistula in the left arm which is non-functional at the moment

There are multiple scars in the right upper chest may be for previous access for hemodialysis

He/She also has evidence of gum-hypertrophy

On examination of the abdomen:

There is a scar in the right iliac fossa which overlies a firm,smooth and non-tender mass which is dull to percussion and there is no bruit over it.

However he/She does not have evidence of:

Uremia

Pallor and

Fluid overload

So based on this:

My most likely diagnosis is End stage Renal disease and

The current mode of Renal replacement therapy is Renal Transplant which is functioning very well

The underlying cause seems to be Diabetes Mellitus

And He/She also has side effects related to steroids use and Immuno-suppressive therapy

## Adult Polycystic Kidney Disease (APKD)

I would like to complete my examination by:

Taking detailed history

Check the observation chart and blood pressure

Dip the urine for hematuria and Proteinuria

I have examined this Gentleman/Lady:

He/She has:

Distended abdomen with fullness in the flanks

But soft and non-tender

There are bilateral flank masses with irregular surface, ballotable and I can get above it

They are resonant to percussion

There is no bruit or rub over it

There is also no evidence of Uremia or fluid overload

So based on this:

My diagnosis is Bilateral Polycystic Kidneys

## Mass in the abdomen (Origin from stomach)

I would like to complete my examination by:

Taking detailed history

Check the observation charts

Examine the external genitalia and hernial orifices

And do the Digital rectal examination (If melena etc)

I have examined this Gentleman/Lady,

He/She is:

Cachectic

With evidence of:

Clubbing

Pallor (Jaundice)

Lymphadenopathy (Site,size,shape,number,tender or not,discrete or matted)

On examination of the abdomen:

It is asymmetrical

There is a mass in the left hypochondrium which is hard with ill defined borders and tender

It is dull to percussion

Not fixed to the abdominal wall

Not ballot-able

Non-pulsatile

Does not move with respiration

There is no bruit or bowel sounds audible over it and

I could get above it

So based on this my most likely diagnosis is mass originating from Pancreas or Stomach









